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The Toxic Effects of Diphenylhydantoin: A Report of 23 Cases

FREDERICK W. FLOYD, M.D.*

Diphenylhydantoin sodium (Dilantin®) was introduced as an anticonvulsant by Merritt and Putnam in 1938.[1] The structure of this drug is analogous to that of the barbiturates, and although the method of action has not been fully determined as yet, its beneficial effects have earned for it a secure place in the treatment of epilepsy, particularly of the grand mal type.

Toxic reactions have been reported in as high as 15 per cent of patients; these are mild and usually consist of lethargy, ataxia, anorexia, nystagmus, and slurring of speech.[2] Thus all reactions which are related to the dose of the drug may be considered toxic in nature; the most common of these is cerebellar incoordination.[3] Other side effects commonly associated with prolonged administration of the drug or attributed to a drug allergy include gingival hyperplasia, dermatitis, purpuric reactions, macrocytic anemia, and a group of "endocrine" disorders involving hypertrichosis and brown pigmentation of the skin.[2, 3]

It is the purpose of this paper to report the toxic effects seen in 23 children. In most instances these were due to an ingestion of an excessive amount; in some cases an overdose had been given; and rarely, therapeutic dosage schedules were followed.

Case Material

During the five year period beginning January 1, 1955 and ending December 31, 1959, there was a total of 55,267 admissions to this hospital. Of these, 1124 admissions were occasioned by poisoning, and of the latter, twenty-three, or 2 per cent, were due to the toxic effects of diphenylhydantoin (table 1).

Twenty of these children were of the Negro race, three were white; there were 12 girls and 11 boys. Patient ages ranged from 1 8/12 years to 8 6/12

* Assistant Chief Resident, Children's Hospital.

TABLE 1

Incidence of Dilantin Intoxication, Children's Hospital 1955–59

	Total Hospital Admissions	Total Poisoning	Dilantin Poisoning
1955	9,399	206	5
1956	10,249	230	0
1957	11,612	253	9
1958	12,160	243	4
1959	11,847	192	5
Totals.....	55,267	1,124	23

years with the median being $3\frac{11}{12}$ years. Toxic symptoms were caused by amounts from as little as 50 mg. twice a day for a period of several weeks, to an amount in excess of 1.5 Gm. taken at one time (table 2). Of the 23 children, five had a known history of pica.

This drug is marketed in various product forms; however, of the 14 cases of intentional ingestion of large amounts, thirteen were of the 50 mg. tablet which has a sweet flavor. In one instance, several children "shared" a bottle containing 100 of these candy-like tablets and four were brought to the hospital the evening following ingestion. Three others who attended the "party" were not seen in the Emergency Room until one or two days later when the parents gradually became alarmed about the symptoms.

Eight of the cases resulted following the use of the drug therapeutically. Four of these were given Dilantin® in a dosage within the recommended range for the child's weight. In one child, too large a dosage was prescribed, and in three children the dosage was not known. In six of these eight patients, the drug was administered from four days to three weeks before ataxia became severe; two children had received the medication for four and six months.

One patient fell ill supposedly as the result of an error in filling a prescription, Dilantin being given in place of an antibiotic; "about eight grains" were taken within 48 hours.

The symptoms most frequently encountered were ataxia, lethargy, vomiting, and vertigo (table 3). Stuttering, aphasia, disorientation, hallucinations and a generalized red "spotty" rash were seen only once.

Cerebellar signs ranged from an unsteady gait through an inability to stand, to a point where even head control was lost. Eleven of the children were noted to have hyperactive deep tendon reflexes. A transient Babinski sign, mydriasis, choreiform movements, slurred speech and spasticity were recorded three times. Two patients, the most severely affected, manifested decerebrate rigidity.

TABLE 2

Summary of Patients

Pt.	Adm. Date	Age		Sex	Race	Wt.	Hosp.	Level of Dilantin	Approximate Amount
		yr.	*mo.*			*kg.*	*days*	*(mg./100 ml. of blood)*	
1. G. A.	2/21/55	4	7	F	N	14	3		30 to 40 of the 50 mg. tabs.
2. C. M.	4/14/55	7	3	F	N	18	5		Increase of dosage from 50 mg. t.i.d. to 100 mg. t.i.d. for 3 weeks
3. R. D.	5/18/55	3	11	M	N	16	2		Unknown number of 50 mg. tabs.
4. D. J.	5/19/55	4	0	F	N	15	6		Continuous dosage of 50 mg. t.i.d. for 4 months
5. D. E.	6/11/55	1	8	F	N	12	3		10 of the 50 mg. tabs.
6. V. B.	3/11/57	1	10	F	N	12	5		12 of the 50 mg. tabs.
7. J. C.	6/29/57	6	3	M	N	20	7		Unknown amount for 3 weeks
8. N. G.	8/14/57	2	0	M	N	12	28	4.2	Unknown amount of 100 50 mg. tabs.
9. R. M.	8/14/57	3	2	M	N	12	7	2.8	Unknown amount of 100 50 mg. tabs.
10. K. M.	8/14/57	3	11	F	N	12	7	3.9	Unknown amount of 100 50 mg. tabs.
11. S. M.	8/14/57	2	0	M	N	10	7	6.6	Unknown amount of 100 50 mg. tabs.
12. E. G.	8/15/57	4	2	F	N	13	7	1.9	Unknown amount of 100 50 mg. tabs.
13. L. B.	8/16/57	3	2	F	N	14	7	2.6	Unknown amount of 100 50 mg. tabs.
14. C. R.	8/16/57	4	5	F	N	15	7	4.7	Unknown amount of 100 50 mg. tabs.
15. J. L.	1/18/58	1	11	F	N	10	14		100 mg. (1 tsp.) b.i.d. for 4 days
16. D. D.	6/ 6/58	3	6	M	W	15	4		Error in filling prescription; pt. received "about 8 grains" in 48 hours
17. K. B.	12/ 4/58	5	7	M	W	16	10		Therapeutic (?) dosages for unknown time
18. E. J.	12/20/58	1	11	M	N	12	3		10 of the 50 mg. tabs.
19. D. T.	4/11/59	3	0	M	N	16	4		20 to 25 of the 50 mg. tabs.
20. R. R.	4/27/59	5	1	M	N	15	12		50 mg. b.i.d. for 6 months
21. W. J.	5/13/59	3	1	M	N	14	13		50 mg. (½ tsp) b.i.d. to t.i.d. for 3 weeks
22. G. W.	6/23/59	8	6	F	W	21	11		Unknown amount for 2 weeks
23. D. D.	8/ 7/59	4	9	F	N	14	8	5.0	Unknown number of 50 mg. tabs.

TABLE 3

Presenting Symptoms and Physical Findings of 23 Patients

	No. of Patients
Symptoms	
Ataxia	20
Lethargy	15
Vomiting	6
Dizziness	5
Physical Findings	
Cerebellar Signs	
Ataxia	12
Unable to stand	7
Loss of head control	2
Hyperreflexia	11
Drowsiness	8
Mydriasis	3
Positive Babinski	3
Slurred speech	3
Choreiform movements	3
Spasticity	3
Decerebrate rigidity	2
Convulsion (grand mal)	2
Mental confusion	2
Loss of consciousness	2

The following case abstracts will serve to point out some of the facets of the problem:

Case 6

V. B., a 1 9/12 year old Negro girl was brought to the Outpatient Department on March 11, 1957 because she had been noted to be "dizzy and staggering" for several hours. She had ingested twelve 50 mg. tablets of Dilantin about 12 hours beforehand.

Upon admission, her vital signs were within normal limits but she was alternately crying out and dozing. She could not stand nor even hold her head erect. There was hyperreflexia and a left positive plantar response; the latter persisted for two days.

Oral fluids were encouraged; her cerebellar incoordination receded; and she was discharged, asymptomatic, on the fifth hospital day. Blood levels of Dilantin were not determined.

Case 8

N. G., a 2 year old Negro boy with a known history of pica, was brought to the Outpatient Department August 14, 1957. He was noted to be lethargic and to have a "drunken" walk when he awoke from a nap. An empty bottle which had contained 100 flavored 50 mg. tablets of Dilantin was found nearby. Following gastric lavage he was admitted to the hospital approximately four hours after ingestion. Vital signs were within normal limits, but he was very ataxic and had hyperactive deep tendon

reflexes. Horizontal nystagmus was present, and the pupils were dilated and reacted only sluggishly to light. The ocular fundi were within normal limits.

The level of Dilantin in blood drawn at this time was later reported as 4.2 mg. per 100 ml. of blood.*

The patient developed increasing attacks of decerebrate rigidity and was transferred to another hospital where dialysis was performed using the artificial kidney. He made a slow but complete recovery in the following weeks. He was discharged, completely recovered, about one month after ingestion.

Electroencephalograms two days after admission showed high voltage but a slow activity pattern with little cortical activity. On this basis decerebrate seizures were controlled with intravenous Tridione®; later encephalograms showed a return toward normal.

Case 23

D. D., a 4 9/12 year old Negro girl, was noted to be "dizzy" on August 7, 1959. She was brought to this hospital and gastric lavage was performed because of a strong history of pica and a questionable story of having swallowed some cleaning powder.

She was a very lethargic young girl, with normal vital signs but cerebellar dysfunction so marked that she could not hold up her head. There was no nystagmus and the reflexes were only slightly hyperactive. A sample of blood was drawn for possible diagnostic determinations. About eight hours after admission, the patient's mother called to say that a cousin's bottle of Dilantin was empty.

There was a rapid recovery from the ataxia and the patient was able to walk with a broad-based gait by the second day.

Subsequent laboratory tests showed the blood to contain 5.0 mg. of Dilantin and 0.068 mg. of lead per 100 ml. She was discharged on the eighth hospital day, to be followed in the Pica Clinic.

DISCUSSION

Within two years after the introduction of Dilantin, the first case of acute Dilantin poisoning was reported. Robinson,[4] in 1940, treated a 25 year old man who had ingested 45 capsules of 100 mg. each and had entered into a deep coma within the hour. Nikethamide, strychnine and caffeine sodium benzoate were all given, and he regained consciousness in several hours. He was discharged on the fourth day without any sequelae.

In the following year, Aring and Rosenbaum[5] reported the case of a 19 year old man suffering from epilepsy who took large doses of the drug "for a spree." These were characterized by ". . . a drunk-reeling gait, thick and rapid speech, generalized tremors . . . blurred vision, diplopia . . . severe ataxia, horizontal nystagmus and generalized hyperreflexia." His toxic manifestations had disappeared by the third day. This same picture was seen again in a 42 year old farmer who attempted suicide[6] and in a 4½ year old boy.[7] The latter case is of interest in that the child also experienced hallucinations. The therapy for the majority of these cases came to

* Performed by the Walter Reed Army Institute of Research.

be increasingly clear: intravenous fluids, analeptics as needed, and careful nursing care to prevent aspiration of vomitus or accumulated secretions.

Tichner and Enselberg,[8] in 1951, reported the fourth known fatality due to Dilantin. This was a 16 year old girl who had ingested an unknown amount and died in deep coma despite intravenous fluids and analeptics. At autopsy the brain was found to be diffusely congested, and contained 6 mg. of Dilantin per 100 Gm. of tissue.

Gropper[9] recorded a case in 1956 in which a 29 year old man had been taking 0.1 Gm. of Dilantin three times a day for about six to eight weeks. This caused a febrile course, a "pimply" rash, jaundice, azotemia and a prolonged prothrombin time. Autopsy revealed severe hepatitis, with widespread necrosis of the hepatic cells. This sensitivity reaction, as well as hemorrhagic erythema multiforme[10] and periarteritis nodosa,[11] has been known to terminate fatally.

More recently Moling and Posch[12] reported 2 cases of intoxication due to this drug. One child, $2\frac{9}{12}$ years of age, took 0.9 Gm. in the form of the 50 mg. tablets, and the other boy, age 3 years, ingested 1.5 Gm. in the form of the suspension. Some of the symptoms seen were ataxia, mental confusion, hyperreflexia, dilated pupils and nystagmus. Treatment included intravenous fluids, analeptics and phenobarbital. Both children had an uneventful recovery.

Dill et al.[13] have shown that the highest concentrations of the drug were to be found in the liver and fat; concentrations in the brain were also high, indicating ready passage through the blood-brain barrier. Administration of single oral doses of 400 mg. to six healthy adults produced blood levels of 2 to 5 mg. per 100 ml. in 8 to 12 hours. The blood levels were found to drop off at the rate of 50 per cent in 17 to 24 hours.

Plaa et al.[14] found the mean blood level of Dilantin to be 0.8 mg. per 100 ml. when the mean daily dose administered to epileptic patients was 4.2 mg. per Kg. of body weight. In general, the blood level was found to be proportional to the dosage.

In this series of 23 patients, hydantoin levels were obtained in 8 patients and ranged from 1.9 to 6.6 mg. per 100 ml. of blood.

SUMMARY

Diphenylhydantoin intoxication was diagnosed in 23 children seen at Children's Hospital from 1955 through 1959. Presenting symptoms included lethargy, ataxia, mydriasis and hyperreflexia. In thirteen of the cases there was *intentional* ingestion of a candy-like preparation of the drug.

In the more seriously ill patients, blood levels of diphenylhydantoin ranged from 2.6 mg. to 6.6 mg. per 100 ml.

There were no deaths in this series. Treatment consisted of intravenous fluids, careful nursing care, and the use of caffeine sodium benzoate.

REFERENCES

1. Merritt, H. H., and Putnam, T. J.: Sodium diphenyl hydantoinate in treatment of convulsive disorders, J. A. M. A. 111: 1068, 1938.
2. Goodman, L. S., and Gilman, A.: The Pharmacological Basis of Therapeutics, ed. 2, New York, The Macmillan Company, 1956, p. 181.
3. Bray, P. F.: Diphenylhydantoin (dilantin) after 20 years; a review with re-emphasis by treatment of 84 patients, Pediatrics 23: 151, 1959.
4. Robinson, L. J.: Case of acute poisoning from dilantin sodium with recovery, J. A. M. A. 115: 289, 1940.
5. Aring, C. D., and Rosenbaum, M.: Ingestion of large doses of dilantin sodium, Arch. Neurol. & Psychiat. 45: 265, 1941.
6. Cattan, R., Frumusan, P., and Attal, P.: Intoxication aiguë par la diphyénylhydantoïne, Bull. et mém. Soc. méd. d. hôp. de Paris 63: 346, 1947.
7. Price, W. C., and Frank, M. E.: Accidental acute dilantin poisoning; report of case with complete recovery in nonepileptic child, J. Pediat. 36: 652, 1950.
8. Tichner, J. B., and Enselberg, C. D.: Suicidal dilantin (sodium diphenylhydantoin) poisoning; case report, New England J. Med. 254: 723, 1951.
9. Gropper, A. L.: Diphenylhydantoin sensitivity; report of fatal case with hepatitis and exfoliative dermatitis, New England J. Med. 254: 522, 1956.
10. Ritchie, E. B., and Kolb, W.: Reaction to sodium diphenyl hydantoinate (dilantin sodium); hemorrhagic erythema multiforme terminating fatally, Arch. Dermat. & Syph. 46: 856, 1942.
11. Van Wyk, J. J., and Hoffman, C. R.: Periarteritis nodosa; case of fatal exfoliative dermatitis resulting from "dilantin sodium" sensitization, Arch. Int. Med. 81: 605, 1948.
12. Moling, J. H., and Posch, J. J.: Acute diphenylhydantoin intoxication; report of two cases, Pediatrics 20: 877, 1957.
13. Dill, W. A., Kazenko, A., Wolf, L. M., and Glazko, A. J.: Studies on 5,5′-diphenylhydantoin (dilantin) in animals and man, J. Pharm. Exp. Ther. 118: 270, 1956.
14. Plaa, G. L., Hine, C. H., and Nelson, T. L.: Blood levels of hydantoins and phenobarbital in epileptics, Fed. Proc. 15: 467, 1956.

Suppurative Parotitis in the Newborn

Nicholas P. Haritos, M.D.*

Suppurative parotitis in the newborn is not a rare disease; the incidence varies from 1 in 2,000 to 1 in 10,000.[1, 2] It should be considered in all cases with parotid area swelling. Two cases of infants with this disease are presented together with a discussion of the disease and its management.

* Formerly Assistant Chief Resident, Children's Hospital.

CASE REPORTS

Case 1

K. T., a 2 week old white male infant, was transferred to Children's Hospital with a one day history of swelling over the right parotid gland area.

The infant was born at another hospital; delivery was by elective low forceps following a normal full term gestation. The birth weight was 8 pounds 2 ounces. Ecchymoses were noted over the face and head at the time of delivery; a prothrombin time performed shortly after birth was abnormal. The infant received a vitamin K preparation, and the prothrombin level returned to normal. Respiratory distress was an additional prominent finding immediately after birth, and the infant was placed in an incubator with oxygen and received combined penicillin-streptomycin medication for four days. These antibiotics were then discontinued and intramuscular chloramphenicol was substituted throughout the remainder of the hospital stay. During the first week of life a hyperbilirubinemia developed and regressed spontaneously.

On the day prior to transfer (13 days of age) a swelling was noted over the right parotid gland area. Peripheral blood examination revealed 44,000 leukocytes per cu. mm. with a shift to the left. Two blood cultures were subsequently reported as negative.

On admission to Children's Hospital, the infant did not appear acutely ill, was in no respiratory distress and had a temperature of 99°F., pulse of 146 per minute and respirations of 40 per minute. There was an indurated, warm, erythematous swelling, measuring 3 by 3 cm., over the right parotid gland. Also noted were ecchymoses of the upper and lower eyelids bilaterally, scattered rhonchi over both lung fields, and a liver edge palpable 1.5 cm. below the right costal margin.

The pertinent admission laboratory studies were as follows: a white blood cell count of 27,200 per cu. mm., with 68 per cent segmented cells, 8 per cent bands, 20 per cent lymphocytes, and 4 per cent monocytes; the hemoglobin was 16.6 Gm. per 100 ml.; the hematocrit was 56 per cent. Urinalysis was interpreted to be within normal limits. A throat culture and cultures taken of purulent material from the right Stensen's duct subsequently produced a heavy growth of *Pseudomonas aeruginosa* and a few colonies of *Diplococcus pneumoniae*. A lumbar puncture revealed 14 leukocytes per cu. mm., all of which were lymphocytes, and a protein of 119.5 mg. per 100 ml. Cultures of the spinal fluid and three blood cultures produced no growth. Skull films were reported as negative.

On admission the intramuscular chloramphenicol was continued. On the first hospital day, the infant became febrile for the first time, developing an elevation in temperature to 101°F. with a prompt return to normal. An erythematous swelling was noted over the left parotid gland at that time. Cultures of the material expressed from the left Stensen's duct grew out a pure culture of *Pseudomonas aeruginosa*. On the second hospital day, because of the culture reports, the chloramphenicol was discontinued and procaine penicillin and Aerosporin® (3 mg. per Kg. per 24 hours) were administered intramuscularly. The infant's condition remained stable for the first three days and he was taking feedings well. On the fourth hospital day, however, the infant became lethargic and anorectic and developed another febrile episode lasting 36 hours. At that point, he became comatose and his temperature fell to subnormal levels. He was placed in an incubator with heat and oxygen, but his condition rapidly deteriorated and he expired on the sixth hospital day.

Postmortem examination revealed a left suppurative parotitis, interstitial pneu-

monitis, ulcerative esophagitis, and a nonfunctional cyst of the left adrenal medulla. Blood and lung cultures taken postmortem revealed pure cultures of *Ps. aeruginosa.*

Case 2

P. J. M., a 5 day old Negro female infant, was transferred to Children's Hospital because of bilateral swelling of the parotid glands of one day's duration.

The patient was the product of a 7 month gestation which was uneventful except for vomiting during the last month. Following rupture of the membranes, labor was 1½ hours in length and terminated in an unsterile delivery of a 3 pound 9 ounce infant. Resuscitation was required for an unknown length of time. On the fifth day of life, swelling of both parotid glands was noted, and the infant was transferred to Children's Hospital.

On admission the infant appeared acutely ill and slightly icteric; respirations were irregular. There was a hard, erythematous, tender swelling over each parotid gland, more marked on the right. Purulent material exuded from both Stensen's ducts.

Peripheral blood examination revealed 19,500 leukocytes per cu. mm., with 34 per cent segmented cells, 37 per cent bands, 7 per cent metamyelocytes, 20 per cent lymphocytes, and 2 per cent monocytes. The hemoglobin was 22.9 Gm. per 100 ml., and the hematocrit was 66 per cent. Culture of the purulent material from Stensen's duct grew hemolytic *Staphylococcus aureus,* hemolytic *Staphylococcus albus,* and *Aerobacter aerogenes.* A blood culture produced a growth of *Escherichia coli,* which was felt to be a contaminant.

Intravenous fluids, chloramphenicol, and aqueous penicillin were administered. Warm, wet compresses were applied over the parotid areas. The patient's condition deteriorated and death ensued on the second hospital day.

Postmortem examination revealed a bilateral suppurative parotitis.

DISCUSSION

The clinical manifestations of suppurative parotitis in the newborn are usually typical, and are demonstrated in the cases reported here as well as those by other writers.[2-4] The disease usually begins with an unexplained febrile episode beginning between the fourth and twelfth day of life, most commonly on the seventh or eighth day. This is followed within 24 hours by swelling of one or both of the parotid glands, with heat and erythema of the overlying skin area. The disease is usually unilateral, but can be bilateral.[5] The area involved may be over the cheek, in front of the ear, at the angle of the mandible or below the horizontal ramus of the mandible. The swelling is usually followed by fluctuation of this region, and at this point purulent material exudes from or can be expressed from Stensen's ducts; this makes the diagnosis a certainty. The stomata of Stensen's ducts are reddened and dilated. Following the initial febrile episode, the patient may continue to be febrile, become afebrile, or develop subnormal temperatures. The latter two are more common in debilitated infants. Anorexia, weight loss, and listlessness are almost always associated.

There is almost always a leukocytosis with a shift to the left; however, the white count may be normal or even depressed. Cultures of the purulent material obtained hopefully produce a growth of the offending organism.

Numerous predisposing factors have been incriminated. These include prematurity, poor general condition of the infant, concurrent infections, puerperal disease, nipple fissures, contaminated nipples, stomatitis, deficient function of not yet fully developed salivary glands, too acid or neutral pH of the saliva, local trauma to the mouth due to brow or face presentation, and forceful wiping of the mouth or overzealous use of suction apparatus.[2, 4, 6, 7]

Approximately 35 to 40 per cent of cases occur in the premature infant.[2, 3] Although all the factors mentioned have been found to occur in some of the cases in the literature, there have been others reported in which none of these has been mentioned;[3] this may be only because all factors were not carefully looked for and considered.

Etiologic agents which have been isolated include hemolytic *Staphylococcus aureus, Escherichia coli, Candida albicans, Bacillus alcaligenes, Diplococcus pneumoniae*, several strains of streptococci, and, with the case reports in this paper, *Pseudomonas aeruginosa*. Hemolytic *Staphylococcus aureus* is the most common offender,[6] which is in keeping with the evidence provided by several investigators that this is the predominating organism in the nasopharynx of the neonate.[8, 9]

Infection may occur by either hematogenous, lymphatic, or retrograde (via Stensen's duct) routes or any combination of these.[4, 10-13]

Berndt, Buck and Buxton,[10] in 1931, using dogs as experimental animals, were able to produce a suppurative parotitis by injecting bacteria into Stensen's duct or into the nutrient artery of the parotid gland. These investigators believed the former to be the easier method. They then compared the pathologic findings produced by each mode of infection. The following is a summary of their results:

Ductogenous	*Hematogenous*
1. Distension of ducts with purulent exudate	1. Purulent exudate in the ducts was less marked
2. Large ducts involved more than smaller ducts	2. Large ducts relatively unchanged; small radicals chiefly involved
3. Extensive destruction of duct epithelium	3. Ductal epithelium intact or occasionally hyperplastic
4. Periductal reaction more prominent where duct wall was broken	4. Periductal reaction more uniform in distribution
5. Periductal cell reaction chiefly neutrophilic	5. Periductal reaction chiefly mononuclear
6. No change in blood vessels	6. Swelling and hyperplasia of arteriolar endothelium with occasional beginning annular thrombus formation

Plewka[4] believed that by histologic analysis, most of his 52 reported

cases were of the ascending ductal type. Parmelee[5] states that the condition is "probably due to infection extending from the mouth to the glands by way of their secretory ducts."

Complications which may occur are involvement of other salivary glands and/or cervical glands, a salivary fistula, facial palsy due to involvement of the seventh cranial nerve, mediastinitis resulting from pus dissecting down the carotid sheath, rupture into the external auditory canal, and septicemia.[4, 14] Involvement of other salivary glands is the most common complication. Because of the possibility of rupture into the external auditory canal, purulent drainage from this orifice should be kept in mind as a presenting finding in this disease.[14]

The differential diagnosis includes all causes of enlargement of the parotid area in the early period of life. This would include epidemic parotitis (rare), cytomegalic inclusion disease, necrosis of the buccal fat pad, and a wide variety of parotid tumors.

The method of treatment of this disease can be divided into two main types; medical and surgical. Both types, however, are usually necessary in each case.[2, 3, 6, 7]

The medical treatment involves primarily the use of antibiotics. The causative organism and its antibiotic sensitivity should be determined and the antibiotic chosen accordingly. Maintaining the general condition of the infant by proper attention to his nutritional needs is another very important consideration. This may be done either by gavage feedings or by the use of intravenous fluids.

The surgical treatment consists of an incision and drainage of the gland when fluctuation has occurred.[2, 4, 6] Reisman and Fischer[13] have stated that surgical intervention is a necessity when suppuration has occurred. The incision is made obliquely below the angle of the mandible.[14] A probe is then inserted and all loculations are opened. Finally, a drain is left in place for at least 48 hours. Possible complications following surgery are seventh cranial nerve injury and salivary fistula formation. To avoid the first of these, Reisman and Fischer[13] prefer to make a vertical rather than an oblique incision.

Neuhauser and Ferris[12] have proposed the use of x-ray therapy to avoid all surgery and its complications. They have advised a dose of 125 roentgens on the first day, to be repeated daily for four days if the desired effect is not obtained by the initial treatment. If there is no response to this schedule, they believe that more irradiation would probably be of no value. These authors reported 3 cases in the newborn period treated with x-ray and feel that the duration of illness was significantly shortened.

A factor which must be borne in mind is that of prevention, since iatrogenic and "nursogenic" causes are listed among the contributing conditions.

All undue trauma to the infant's mouth, including strenuous wiping of the mouth, and overzealous aspiration of the mouth and upper respiratory tree should be avoided. Other more obvious modes of prevention are the avoidance of contaminated nipples and the discontinuance of breast feeding by a mother with a significant systemic or breast infection.

The reported prognosis of this disease ranges widely, depending on the author, and varies from over 50 per cent mortality to "good;"[2, 5] the prognosis is felt, however, to be poorer in premature and generally debilitated infants.

SUMMARY

Two infants with acute suppurative parotitis occurring in the neonatal period are presented. The etiologic agents were hemolytic *Staphylococcus aureus* and *Pseudomonas aeruginosa*, respectively. The clinical manifestations are presented, together with several predisposing factors, of which prematurity is the most common. *Staphylococcus aureus* is the most likely offending organism. The complications and differential diagnosis are also presented. The treatment of this disease involves the use of antibiotics and surgical incision and drainage. X-ray therapy has also been suggested. The prognosis is variable, but poorer in premature and debilitated infants.

REFERENCES

1. Campbell, W. A. B.: Purulent parotitis in newborn; report of case, Lancet **2**: 386, 1951.
2. Sanford, H. N., and Shmigelsky, Irene: Purulent parotitis in the newborn, J. Pediat. **26**: 149, 1945.
3. Durand J.: Practice of Pediatrics, Hagerstown, Md., W. F. Prior, 1959, vol. 3, chap. 2, p. 14.
4. Plewka, W.: Arch. Kinderh. **69**: 279, 1921, as cited by Elterich (ref. 11).
5. Parmelee, A. H.: Management of the Newborn, ed. 2, Chicago, Year Book Publishers, Inc., 1959, p. 330.
6. Richman, J. D., Massler, M., and Schour, I.: Textbook of Pediatrics, ed. 7, Philadelphia, The W. B. Saunders Company, 1959, p. 637.
7. Shulman, B. H.: Acute suppurative infections of salivary glands in newborn, A. M. A. Am. J. Dis. Child. **80**: 413, 1950.
8. Smith, J. W., and Bloomfield, A. L.: Development of aerobic bacterial flora of throat in newborn babies, J. Pediat. **36**: 51, 1950.
9. Torrey, J. C., and Reese, Martha K.: Initial aerobic flora of newborn premature infants, nature source and relation to ultraviolet irradiation and face masks, A. M. A. Am. J. Dis Child. **67**: 89, 1944.
10. Berndt, A. L., Buck, R., and Buxton, R. von L.: Pathogenesis of acute suppurative parotitis. Experimental Study, Am. J. M. Sc. **182**: 639, 1931.
11. Elterich, T. O.: Purulent parotitis in the newborn; case report and review of the literature, J. Pediat. **3**: 761, 1933.
12. Neuhauser, E. B. D., and Ferris, B. G., Jr.: The treatment of acute suppurative parotitis in infants, J. Pediat. **27**: 589, 1945.

13. Reisman, H. A., and Fischer, Golda: Acute suppurative parotitis in a newborn premature infant, A. M. A. Am. J. Dis. Child. 71: 387, 1946.
14. White, R. H. R.: Acute suppurative parotitis in the newborn, Brit. Med. J. 1: 1232, 1952.

Primary Tracheal Papilloma: A Case Report

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In a complete review of the literature through 1953, Gilbert et al.[1] collected only 43 cases of primary tracheal tumors occurring in children. During the period 1953–1959 a single case report[2] was added to the English literature. The purpose of this paper is to record the first instance of a tracheal neoplasm diagnosed at Children's Hospital and to review briefly the symptomatology and problem involved in diagnosis.

CASE REPORT

This 9 year old Negro boy was admitted to Children's Hospital on December 30, 1959 with a 3 month history of progressive respiratory distress. The patient had been seen for the first time in the Medical Clinic on October 8, 1959. He had been well until two weeks prior to the initial visit when wheezing respirations and cough insidiously developed. There was no history of previous respiratory distress and no history of allergy or foreign body aspiration. On examination, slight expiratory prolongation and wheeze were noted. There was no stridor or retraction. A diagnosis of mild bronchial asthma was made. An oral bronchodilator was prescribed and the patient instructed to return. Two days later, slight subjective improvement was reported following the use of the prescribed medication. One month before admission, his symptoms intensified and he complained now of "shortness of breath" aggravated by recumbency. Three weeks before admission, he was examined in the Allergy Clinic which concurred with a diagnosis of asthma. One week prior to admission, his cough became productive of thick white sputum, particularly during the night when coughing spells were prone to occur. On examination the day of admission, the patient was noted to have inspiratory stridor and retraction which prompted radiologic examination of the larynx and trachea and emergency hospital admission.

History of past illness and family history were noncontributory.

Physical examination on admission to the hospital revealed an afebrile, thin, well developed Negro boy with marked inspiratory stridor but a slow respiratory rate (16 per minute) and a prolonged inspiratory phase. There was no cyanosis. The oropharynx appeared normal. No masses were palpable in the neck. There was deep retraction of the suprasternal notch on inspiration. The lungs were clear except for transmitted sounds. The heart sounds were of good quality and no murmurs were

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noted; the heart rate was 100 per minute. The remainder of the physical examination was noncontributory.

The blood count and urinalysis were within normal limits. A lateral x-ray of the neck demonstrated a dense shadow attached to the anterior tracheal wall at the level of the sixth cervical vertebra, almost completely occluding the tracheal lumen (fig. 1).

The patient was admitted to the surgical service and scheduled for bronchoscopy; prior to this a tracheotomy was performed. Immediate relief was established with insertion of the tracheotomy tube. The tumor, which bled on contact with the exploring bronchoscope, was easily visualized. A number of tumor pieces were removed with the biopsy forceps. The pathological report of the biopsied specimen was benign squamous papilloma.

FIG. 1. Lateral view of the neck clearly showing the obstructing tracheal intralumenal mass at level of sixth cervical vertebra.

A repeat bronchoscopic examination was performed nine days later with removal of more tumor fragments. The postoperative course was uneventful. There was no recurrence of dyspnea. The patient was subsequently discharged, asymptomatic, with the tracheotomy tube in place but occluded.

DISCUSSION

The papilloma is by far the commonest primary tracheal tumor occurring in children, comprising greater than 50 per cent of the reported cases. Other benign forms encountered are the fibroma, angioma, and chondro-osteoma. Three cases of tracheal sarcomas have been recorded and represent the only malignant tumors encountered.[1]

Clinical Features

There is no age or sex selectivity in childhood. Symptoms are mechanically produced and are related to tumor size and the degree of obstruction. The site of tumor formation, size, and the type of attachment are directly responsible for the signs produced. A pedunculated tumor located near the adjacent larynx or bronchi is capable of prolapse and causing obstructive findings whereas a tumor with a broad base attachment accounts for fewer symptoms. Wheeze, the most common symptom of tracheal tumors, was described initially by Jackson[3] as an "asthmatoid wheeze" heard at the open mouth; the sound is caused by a column of air set in vibration by passing through a narrowed lumen. The wheeze generally precedes the onset of dyspnea. Cough is frequently an early manifestation, and is initially dry in character until obstruction with retention of secretions and often infection create a productive return. Stridor, retraction, and dyspnea are obviously directly proportional to the degree of occlusion and narrowing of the airway. Rarely, hemoptysis, hoarseness or aphonia may occur.

Diagnosis is by endoscopic examination. This procedure is indicated in a child of any age whose clinical features are compatible with unexplained high respiratory obstruction not of an acute infectious nature. In occasional instances the tumor mass may be demonstrated roentgenologically. This procedure should precede endoscopy when the severity of symptoms does not preclude its use. Tomography is a valuable aid for exact determination of tumor location and extent.

Differential Diagnosis

To be included in the list of differential diagnoses are all of the conditions producing high respiratory obstruction typically inspiratory in character. Congenital laryngeal stridor, congenital laryngeal cysts, and laryngeal papillomas are diagnosed by laryngoscopy. Papillomas of the larynx usually occur in females. Their primary site of origin is the larynx though they may grow down into the trachea. They tend to disappear spontaneously at

puberty whereas papillomas of the trachea remain stationary or grow larger in adult life. The symptoms of laryngeal involvement are primarily hoarseness and dyspnea. Laryngeal or tracheal foreign bodies, occlusion by vascular anomalies, compression by adjacent tumor or thyroid enlargement may all offer problems in differential diagnosis. Finally, tracheal tumors may masquerade as bronchial asthma early in their development. The expiratory wheeze, mild dyspnea and much greater incidence of asthma may satisfy the diagnosis of an "asthmatoid syndrome." The insidious course, and the constant and progressive symptomatology in the absence of the typical episodes of acute expiratory distress characteristic of asthma dictate the necessity for investigating the upper respiratory tree.

Treatment

Removal of benign tumors through the bronchoscope is often successful.[4] With more extensive tumors an open procedure may be necessary with concurrent reconstruction of the tracheal wall. It may be necessary before carrying out either procedure to perform a preliminary tracheotomy, thus overcoming dyspnea and providing an easy route for administration of intratracheal anesthesia. Malignant tumors of the trachea amenable to surgery require open operation with removal of the mass and underlying cartilaginous rings followed by a tracheoplastic repair. Frequent follow-up examinations are necessary because of the recurrent nature of tracheal tumors.

SUMMARY

A case of a primary tracheal tumor occurring in a child is reported. Its possible confusion with asthma is emphasized.

REFERENCES

1. Gilbert, J. G., Mažzarella, L. A., Feit, L. J.: Primary tracheal tumors in the infant and adult, A. M. A. Arch. Otolaryng. 58: 1, 1953.
2. Death during general anesthesia for bronchoscopy in a child, Clinical Anaesthesia Conference, New York J. Med. 56: 1832, 1956.
3. Jackson, C.: Tumors of the trachea, South Med. & Surg. 5: 263, 1936.
4. Moersch, H. J., Clagett, O. T., and Ellis, F. H., Jr.: Tumors of the trachea, M. Clin. N. America 38: 1091, (July) 1954.

Allergic Nasal Obstruction in the Child*

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Persistent nasal obstruction due to allergy in the child represents a paramount problem both to the parent and to the physician. The nasal involvement of the allergic child may be of two main varieties, one of which has been termed recurrent and the other perennial. Distinction between the two is primarily by history. In the recurrent group the child gives a history of repeated episodes of nasal stuffiness and discharge, sneezing, or itching; a postnasal drip and a hacking cough may also occur. This particular episode may rapidly change with the advent of secondary infection and present with low grade fever and change in character of the discharge from mucoid to purulent, lasting from several days to one to two weeks. Characteristic of this disorder, however, are the recurrent episodes: the child goes from one "cold" to another, is free of symptoms for a week, and goes back to school only to have symptoms recur throughout the winter months.

Perennial allergic rhinitis, on the other hand, causes almost constant nasal obstruction. The child is a notorious mouth breather. At night he lies in bed with mouth open, frequently snoring loudly. A cough may or may not be present. There rarely is much nasal discharge; instead his mother complains that he will not blow his nose. This is not surprising since there is very little mucus present; the nose is completely occluded by soft tissue.

These conditions must first be differentiated from those caused by infection; this may be no easy matter. However, the physical findings most often help make the diagnosis. In primary nasal infections, the mucous membranes are usually reddened, there is either mucopurulent or purulent nasal discharge, and nasal smears contain mostly polymorphonuclear cells, with eosinophils few or absent. There is no other sign of allergy in the individual patient and very rarely is there any history of allergy in the family. Sinus involvement is not common. There is no evidence of wheezing and nothing in the x-ray to indicate persistent or recurring pulmonary involvement. Skin tests are usually negative.

In the patient with nasal allergy, either recurrent or perennial, the mucous membranes are pale, shiny and edematous; the mucus to be discharged is much more watery. Nasal smears will show eosinophils, usually 10 per

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cent or more, often as high as 100 per cent. Other signs of allergy are usually present if one searches for them. Characteristically, during acute exacerbations the breath sounds are somewhat wheezy, and if the child inspires and expires very deeply, fine crackling rales may be heard at the bases of the lungs. If these children have sinusitis, it is usually of the hyperplastic or mucous membrane thickening type rather than that due to actual purulent infection. Roentgenograms usually show increased bronchial markings as a result of the constant postnasal discharge and cough. Skin tests are usually positive.

It is also necessary to distinguish between nasal allergy and adenoid enlargement. Many a child with this same history of difficulty in breathing has a wide-open anterior nasal passage; the turbinates are in their proper positions and are not edematous or red, and there is no mucus in the nasal passages. Nasal obstruction in these children is further posteriorly and usually due to adenoid enlargement.

The nasal smear is a simple office procedure, and is performed by collecting nasal mucus on a small swab moistened with saline. The swab is merely placed, or perhaps very gently rotated, on the floor of the nose and allowed to stay until enough mucus can collect; the mucus thus obtained is rolled on a clean glass slide. This can be air dried and stained in the customary fashion with Wright's stain. For sharper distinction the slide may then be decolorized with 95 per cent alcohol for one to two minutes, washed, and briefly counterstained with methylene blue; this provides a sharp contrast between eosinophils and polymorphonuclear cells of other varieties. Greater than 10 per cent eosinophils usually indicates an allergic problem; their absence on a given examination is not too helpful, but repeated negative tests are fairly conclusive.

Nasal polyps in children are almost pathognomonic of allergy. These are not treated by removal (unless necessary because of size), but by treatment of the underlying cause; unless this is done they are very apt to recur.

Sinusitis in the child is usually of the allergic type. Children with sinusitis generally have thickened sinus membranes which produce a mucoid discharge; purulent discharge is rare. The sinus membranes take part in the same allergic reaction as the nasal membranes, and when the nasal mucosa clears under treatment the sinus membranes usually follow suit somewhat later.

In children with allergic nasal obstruction should the tonsils and adenoids be removed? Two statements can be made with little fear of contradiction. First, any child who has a history of having had his adenoids out more than once is automatically classified allergic until proven otherwise. Second, the untreated allergic child who has had his adenoids and tonsils removed may have his first attack of bronchial asthma in a matter of a few weeks or

months following this operation. These two facts have led us to feel that caution in the removal of adenoids and tonsils in the allergic child is extremely important. The proper approach in a child with coincidental nasal allergy and tonsil and adenoid enlargement or infection is to treat the allergy first. Very often a period of management of three to six months is sufficient to control the allergic manifestations, and the operation can be performed with impunity. On the other hand, in some instances with proper treatment the necessity for operation may disappear.

Etiologic diagnosis may or may not require the help of an allergist but there is no reason why the pediatrician cannot attempt it first. Nasal allergies are largely due to inhalant materials; these are usually the common ones found about the house. Occasionally pollens are involved, but typical hay fever in a child is much less frequent. Foods, unless the history indicates it, are not often a cause of nasal allergy. In our experience, the number of children in whom foods have played a role have been few in number by comparison with those due to environmental allergens. Probably the one best initial routine is to try to eliminate the usual contact materials in the child's bedroom. Next, one may try symptomatic treatment, such as the antihistaminics. If a good response is obtained, this may be enough, but if recurrences do take place, one may wish to go further into the etiologic diagnosis by skin testing and, if indicated, desensitization.

The Editor's Column

Specialization in Medical Practice: Is There a Place for Pediatric Surgery?

Reflection upon the present position of the practice of medicine and its professional organization is always salutary. It is necessary periodically to stand back from the minutiae of everyday practice and examine the over-all plan. Dr. May's *Wall Memorial Lecture*[1] in May 1960, is a presentation of his reflection on pediatrics in this country. It raises the larger question of the definition of a specialty in medicine. This lecture and the panel discussion which followed[2] illustrate the divergent views. These views might be roughly epitomized by saying, on the one hand, that a specialist is one who diligently seeks to know more and more about less and less; on the other, that a specialist is one who dispenses specially acquired knowledge to as many as may need it. Both views are correct if put in the right context; the problem is whether they can be, or should be, combined. Here again, there is

no "right" or "wrong" answer. As in so many discussions of this type it is necessary to say "it all depends." In this instance, it depends upon the degree of specialization being discussed.

In medical practice, it is customary to make a distinction between "general practitioners" and "specialists." This is an archaic division and means very little. General practitioners have their own special type of practice requiring special skills. Members of the remainder of the medical profession describe themselves as "specialists," but some are very general specialists and some very specialized specialists. Two factors tend to make this division; one is inherent in the particular specialty and the other in the specialist himself. The discipline of the specialty may lead to the development of superspecialties necessitating long training to acquire a unique skill and requiring special apparatus, facilities, or teams to provide the special service. The personality of the specialist may lead either into general proficiency over his whole field, or into paths which will allow him to acquire special knowledge within his specialty. The position of the research worker and the teacher can be seen to be divided in similar ways. The general practitioner can make, and in the past has made, clinical observations as significant and valuable as the most esoteric laboratory worker. On the other hand, certain types of research require the time and special facilities which only workers with subsidies or grants at special institutions can undertake.

It would be invidious and foolhardy to attempt to set a relative value on the work of the superspecialist versus that of the specialist or general practitioner, or of the research worker versus that of the clinician with a part-time interest in research and teaching. Each has his essential place, and each contributes an irreplaceable service to medicine as a whole. It is only when the clinician attempts basic research for which he has neither the time, the facilities, nor the training that the combination of research clinician fails. Similarly the laboratory worker seeking knowledge for its own sake usually has neither the time nor the inclination to be a good clinician and teacher. Yet, without him, the clinician could never have developed the control over disease which is now taken for granted. The specialty of "pediatrics" should not be confined to one approach to practice.

There is a need for three types of pediatricians. There is a need for the pediatrician with special qualifications for supervising the care of children in health and disease, for the practicing of what could be called "known pediatrics." There is a need for the research clinician to staff centers where unknown clinical problems can be investigated. But there is also a need for the man who seeks after knowledge in the pediatric sphere for its own sake. This is not to say that one individual cannot combine at least two of these disciplines, but it is unlikely that he will do either quite as well if he does. The practice of "known pediatrics" entails, in addition to the immediate

care of patients, the obligation to teach better care to others. A specialty should never set itself up as a closed shop. It should be prepared both to prove itself by providing either a superior service or a hitherto unavailable service and to encourage others to provide this service. It would be as wrong to suggest that the pediatrician should discourage the general practitioner from treating children as to suggest that he should only see children as a consultant. The pediatrician should, however, set a standard of child care in his area which the general practitioner would have to emulate. And he should serve in a pediatric consultative capacity to the general practitioner.

With these thoughts in mind regarding the pediatrician and his specialty, it seems fair to discuss the justification of pediatric surgery as a specialty. The establishing of such a specialty gives rise to two problems, a general and a particular. The general problem is that there remains a tendency to regard, from a surgical point of view, children as miniature adults. In the majority of cases children come to surgery with the same conditions as adults, i.e., inguinal herniae and acute appendicitis. General surgeons, therefore, can deal very adequately with the majority of the children on the surgical service. This, though true, becomes progressively less so as one descends the age scale; it is not applicable to the newborn and may be lethal to the premature. The particular problem arises because of the subdivisions of surgery into regional and system subspecialties. The fact that the pediatric surgeon tends to cross the barriers of these subspecialties may have distinct advantages. Witness the valuable contributions made by the pediatric surgeon to the subspecialties of surgery which have subsequently been profitably applied to adults, in such fields as cardiovascular surgery, plastic surgery and abdominal surgery.

It is in the premature and newborn infants that the need for this "specialty" becomes most apparent. The average general surgeon and his subspecialist colleague do not acquire in the course of their ordinary training any special knowledge of, or any appreciable exposure to, the unique surgical problems which arise in the newborn. A thorough familiarity with the various types of congenital defects and neonatal accidents, anatomic and physiologic, with their natural history, is fundamental to rational treatment. Detailed knowledge of the tried methods, both successful and unsuccessful, for dealing with these peculiar problems is mandatory. Up-to-date information regarding the work, and particularly the results, of others in the same field is also essential, for without continuous self-criticism any specialty will settle into stagnant mediocrity.

In an earlier paragraph, it was pointed out that some specialties justify themselves because of the special facilities and team work which they require. This is particularly true of the surgery of the newborn and very young infant. In order to achieve success in this field the most exacting, detailed

supervision of preoperative, anesthetic and postoperative care is required, quite apart from any "surgical" knowledge as to when and how to operate. This care must be continuous and requires, not only time-consuming medical supervision, but nursing vigilance of a very high order. This cannot be expected of an ordinary hospital. Unless a special unit is provided for the surgical care of infants with a disproportionately high number of skilled nurses in continuous attendance, unless the physical arrangement is such that all the infants are observed by a skilled attendant at all times, unless the physical environment of each infant is constantly controlled, the results will not be as good as they could be.

It is pertinent to examine the actual extent to which such a unit could be expected to be used and how much actual benefit to the community it would be. Such a study was made in Liverpool, England, by Rickham[3] and Forshall and Rickham.[4] This study is relevant because the Liverpool and District of Columbia areas are comparable in population, provisions of hospitals and environs. In the Liverpool area, in 1949, at least 75 infants with urgent surgical conditions were born; 21 never reached the hospital and died; 54 reached one of 14 different hospitals in the area and were operated upon. The mortality rate was 66 per cent. In 1953, a small neonatal surgical unit of eight beds and two to six incubators was established in an attempt to concentrate these cases and improve the results. During the first 6 years this unit was functioning, 813 infants were admitted, with an over-all mortality of 24 per cent. One hundred fifty of these infants were found to have a medical condition not requiring surgery, but the remaining 663 were operated on, with a 26.7 per cent mortality. These figures show that within four to five years, coincidentally with the organization of a well publicized neonatal surgical unit, the mortality for newborns with acute surgical emergencies dropped from approximately 72 to 26.7 per cent. In the Liverpool report there is a table giving details of the disorders treated; some of the figures are worth quoting as an example of what can be achieved. There were 27 cases of intestinal obstruction (excluding meconium ileus) in premature babies who had no other malformation; only three died. There were 83 cases of "esophageal atresia" (presumably including those with a fistula); forty-three died, but forty-six were premature and thirty had some other abnormality; only 4 out of 26 full term babies without other abnormalities died.

Bonham Carter and Waterston,[5] in reporting their work to the British Association of Pediatric Surgeons in 1960, stated that the mortality for infants under 3 months of age admitted with cardiac failure was 46 per cent when medical therapy alone was offered. In their special unit at Great Ormond Street Hospital, they began repairing cardiovascular anomalies in infants under 3 months of age, with a reduction of over-all mortality to 26 per cent. This was a preliminary report, and they remained, at that time, far from satisfied with their methods.

Similar results could be achieved in the District of Columbia if a special unit for neonatal surgery were established. Such a unit is expensive to set up, to staff and to operate. It is necessary to have continuous direction by one individual and would not work under a system of rotating chiefs. It is not necessary that all surgery be performed by the director of the unit, who should, however, be available to take charge of any case referred to him or to act as a consultant to his colleagues. Nevertheless, at all times he should supervise the special care in the unit. In this way he would truly fulfill the function of a specialist; to set a standard, and to share his knowledge.

The National Foundation is particularly interested in congenital malformations. Almost all newborn infants requiring urgent surgery fall into this general classification. It is possible that this foundation would give favorable consideration to helping Children's Hospital or any hospital establish such a unit if the Medical Board and the Board of Trustees would support a surgeon applying for a grant to do this. Such a unit would fit well into any scheme for establishing Children's Hospital as a regional, diagnostic and treatment center. It is to be hoped that those interested in the future of Children's Hospital will give serious thought to the advisability of establishing a neonatal surgical unit, and that pediatricians will give encouragement to those who are working toward this goal.

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REFERENCES

1. MAY, C. D.: The future of pediatricians as medical specialists in the United States, Clin. Proc. Child. Hosp. **16**: 317, 1960.
2. ANDERSON, W. S., AND OTHERS: Panel discussion: what is the new pediatrics? Clin. Proc. Child. Hosp. **16**: 328, 1960.
3. RICKHAM, P. P.: Neonatal surgery; early treatment of congenital malformations, Lancet **1**: 332, 1952.
4. FORSHALL, I., AND RICKHAM, P. P.: Experience of a neonatal surgical unit, Lancet **2**: 751, 1960.
5. BONHAM CARTER, R., AND WATERSTON, D.: Personal communication to the author, 1960.

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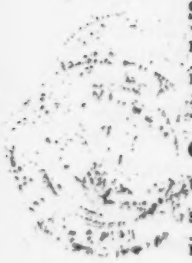

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1. Kane, S.: Am. Pract. & Digest Treat. *8*:65 (Jan.) 1957.

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